

POSTOPERATIVE SYNERGISTIC GANGRENE ON THE ANTERIOR ABDOMINAL WALL – REPORT OF A CASE

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ABSTRACT

The management of a 23 year old boy who postoperatively developed severe anterior abdominal wall infection diagnosed as postoperative synergistic gangrene (Meleney disease) following laparotomy for penetrating abdominal injury is presented. The patient was managed with early aggressive debridement in the theatre, repeated bedside debridement over several weeks and intravenous antibiotics. His wound healed by secondary intention and he was discharged after 46 days on admission.

KEYWORDS: postoperative synergistic gangrene, bedside debridement

INTRODUCTION

Postoperative synergistic gangrene is a rare rapidly spreading and relentless destructive subcutaneous lesion first described by Meleney as post operative synergistic gangrene in 1931 (Meleney, 1931). A variety of terms has been used to describe this condition including necrotizing fasciitis, progressive synergistic bacterial gangrene, and hospital gangrene (Wilson, 1952; Gannon, 1994). Differentiation from cellulitis and abscesses is important but difficult in the early phase because of paucity of early signs, and is usually diagnosed because the disease continued to progress in spite of therapy (Wong *et al*, 2003).

The infection usually dissects along fascial planes with extensive necrosis of the fascia and undermining of the surrounding structures. Skin gangrene is due to thrombosis of the nutrient vessels. The disease lies as a clinical entity between cellulitis and myonecrosis with aggressive necrosis of skin and fascia while sparing muscle (Goldberg *et al* 1984). Mortality has been reported to be 21.3% from Singapore and 50% in Ilorin, Nigeria (Wong *et al* 2003; Adigun & Abdulrahman, 2004).

The incidence of necrotizing fasciitis has been on the increase because of increase of immunosuppressed patient with diabetes mellitus, cancer, alcoholism and HIV (Sharkawy *et al* 2004). A case of postoperative gangrene in a previously healthy young man is presented and the challenges we had in managing him is discussed.

CASE REPORT

A 23 year old boy was referred to our center on account of spreading infection in his operative wound. He was involved in an inter-tribal clash and sustained an extensive laceration in the left hypochondrial region from a cutlass. He had immediate laparotomy in the referral center where repair of small bowel laceration was done. On the fourth day post surgery the wound was noticed to be draining brownish fluid and the skin sutures were removed. Two days later, extensive bullae were noticed in the anterior abdominal wall and he was then referred. On examination, the patient was febrile temperature of 38.4 degree Centigrade, tachypnoeic with a respiratory rate of 32 cycles/min with tachycardia of 104/min. Examination of the abdomen showed a transverse left hypochondrial wound and a midline wound without skin sutures with extensive bullae of the skin of the anterior abdominal wall. Both the wounds and bullae were draining brownish foul smelling fluid (Figures 1 & 2).

Investigations done showed a white cell count of 15,700 cells with 92% neutrophils. Packed cell volume was 23%. Electrolytes, urea and creatinine were within normal limits. Urinalysis showed no abnormality. Culture of the wound showed mixed growth of streptococcus and coliform organism. Abdominal ultrasound did not show any intraperitoneal fluid collection or abscesses and plain abdominal X-rays were normal. He was assessed as having postoperative synergistic gangrene.

He was commenced on intravenous fluids – Ringers lactate alternating with 5% dextrose water 3 liters a day, intravenous ceftriaxone 1 gram 12 hourly and metronidazole 500mg 8 hourly. He was transfused with 3 units of blood. He underwent a wound debridement in theatre. It was noticed that there was extensive fascia involvement beneath the skin. All bullae and dead fascia were excised; wound was irrigated with copious saline and dressed. Daily saline dressing was continued and bedside debridement was done four times over the following three weeks. Figure 3 shows appearance of wound after 3 weeks of treatment.

The patient's condition improved and wound healed by secondary intention (Figures 4 & 5). He was discharged home on the 46th day of admission.

DISCUSSION

Postoperative synergistic gangrene is a deadly form of soft tissue infection that has been tagged "Meleney's Minefield" (Wong *et al*, 2006). It has a mortality rate of 34% (range of 6%-76%). Survival depends on early recognition and prompt aggressive debridement along with targeted antibiotic therapy.

Most patients with postoperative synergistic gangrene had pre existing immunosuppressive condition such as diabetes mellitus, chronic renal failure and HIV or are elderly or obese. Our patient was a healthy young man who had not used any hard drugs and the only identifiable risk factor was surgery as reported in 4.5% of cases (Wong *et al* 2003).

Diagnosis of postoperative synergistic gangrene is difficult due to paucity of early cutaneous signs. In the review by Wong *et al* 2003, only 14.6% of their 89 patients were diagnosed at the time of admission (Wong *et al*, 2003). They gave an excellent guide for making early diagnosis of this condition. Diagnosis in this patient was made because of the presence of characteristic bullae in the anterior abdominal wall. Subsequent full-thickness skin and superficial fascia necrosis sparing underlying muscle differentiated it from a clostridial myonecrosis. Invading organisms were identified from culture of wound swab. Although culture of wound biopsies could not be done for logistic reasons, culture of wound biopsies taken from the spreading periphery of the necrotizing infection gives higher yield. Associated systemic conditions including diabetes mellitus, HIV were ruled out in this case as non-recognition and management of systemic comorbidities may prove inimical to success. The diagnosis was confirmed intra-operative by the presence of foul smelling and dirty serous fluid, pus and grayish necrotic fascia. Other significant findings were the loss of resistance of the deep fascia to blunt dissection and lack of bleeding of the fascia during dissection as described in the literature (Akhtar *et al*, 2010). Plain abdominal xray and abdominal ultrasound showed negative findings. However computerized tomography scan and MRI have been reported to aid early diagnosis (Wyoski *et al* 1998, Schmid *et al* 1997) but their use should never delay operative intervention (Wong *et al* 2003). A high index of clinical suspicion is thus required for early diagnosis and intervention.

The management of postoperative synergistic gangrene is aggressive resuscitation, surgical debridement with excision of all dead tissues and targeted elaboration of broad spectrum antibiotics (Adigun and Abdulrahman 2004). A second look within 24 and 48hrs should be done to assess the need for serial debridement; (Wong *et al* 2003) as occurred in our case. Due to limited financial resources, our case had subsequent serial debridement by bedside under some analgesia. The resultant skin defects contracted adequately and were allowed to heal by secondary intention. Skin grafts/flaps are recommended to cover the extensive defects resulting from debridement, but the financial constraint in this patient precluded skin graft (Adigun and Abdulrahman 2004). The use of negative pressure wound therapy and hyperbaric oxygen therapy in the management of these patients have been reported to improve survival and shorten treatment times (Phelps *et al*, 2006). These modalities are not available in resource limited countries. Therefore management should be based on early recognition, early surgical debridement and broad spectrum antibiotic therapy.

CONCLUSION

We recommend a high index of suspicion for postoperative synergistic wound infection and early radical debridement of suspicious postoperative wound. Repeated debridement can be done safely by the bedside of the patient, thereby reducing overall cost in the treatment of these patients.

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Figure 1 – Appearance at presentation 1



Figure 2 – Appearance at presentation 2



Figure 3 – Appearance at 3rd week of treatment



Figure 4 – Appearance at discharge 1



Figure 5 – Appearance at discharge 2

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